SAMe Deficiency Linked to Development of Alzheimer’s Disease

Scientists at the University of Massachusetts, Lowell, have reported a possible link between deficiency in the naturally occurring chemical, S-adenosyl-methionine (SAMe) and the development of Alzheimer’s disease. Working with mice bred to possess genetic risk factors for the development of dementia, investigators noted that these mice developed oxidative damage leading to cognitive impairment when deprived of the B-vitamin, folic acid.

The mice were found to be deficient in SAMe, which acts as a methyl donor throughout the body, facilitating countless biochemical reactions. When the animals were supplemented with SAMe, neurodegeneration was alleviated. SAMe deficiency promotes overexpression of a protein, presenilin-1, which in turn increases production of amyloid-beta, an aberrant protein directly implicated in the development of Alzheimer’s disease. “These findings directly link nutritional deficiency and genetic risk factors, and support supplementation with [SAMe] for Alzheimer’s therapy,” investigators concluded.1 Subsequent research by the same team confirmed and supported these conclusions.2

—Dale Kiefer

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